Reentrant Tachycardia: Participation of the Distal A-V Conduction System*

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Paroxysmal supraventricular tachycardia (SVT) is commonly produced by two mechanisms: atrial reentry via an A-V nodal pathway or a rapid firing ectopic focus. Studies in the experimental animal and in human beings with sustained SVT have shown that the site for reentry appears to be the upper A-V junction. This report documents an unusual site of reentry in a patient with recurrent SVT. Progressive intraventricular conduction delay with prolongation of the H-V interval resulted in initial conduction delay and then sustained reentry. This strongly suggests that, in man, the distal A-V conducting system may participate in a reentry circuit producing and perpetuating SVT.

Recent work has emphasized that paroxysmal supraventricular tachycardia (SVT) may originate by two mechanisms: a) atrial reentry via an A-V nodal pathway secondary either to premature atrial contractions1 or sinus beats in the presence of A-V dissociation2 and b) rapid firing of an ectopic atrial focus.3 Goldreyer and Damato,4

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Wit et al5 and Janse and colleagues6 in the human being and in isolated cardiac tissue have obtained supporting evidence showing reentry to occur at the A-V node and lower atrial tissues during SVT.

The purpose of this report is to present a case of paroxysmal supraventricular tachycardia with an unusual reentry pathway utilizing, in part, the more distal A-V conduction system.

CASE REPORT

A 62-year-old caucasian woman was admitted to the hospital because of palpitations. These episodes were first noticed when the patient was 11 years old and recurred approximately once a month. During her first pregnancy, she had a prolonged episode of palpitation which resulted in spontaneous abortion. Over the past ten years, the attacks have increased in frequency to three to four per day, and with a duration varying from five minutes to 26 hours. During these episodes, she was weak, vomited, became diaphoretic and occasionally experienced syncope with loss of urine and feces. She has been hospitalized three to four times a year for the past five years. Over the past six months, the arrhythmias have increased in duration and her strength has decreased. Cardioversion has been required on two occasions and, in addition, the patient has been treated with digitalis, quinidine, diphenylhydantoin (Dilantin), and propranolol. She had shortness of breath when walking uphill, but could walk on level ground without dyspnea. Exercise did seem to precipitate her attacks. She was in congestive heart failure and hospitalized for that reason five years ago. Physical examination showed blood pressure of 230/100 mm Hg with an irregular pulse of 64/min.

A slight left ventricular lift was present. A grade 1/6 midystolic murmur was heard at the apex and an S4 gallop was present. The chest x-ray film demonstrated cardiomegaly with interstitial edema. A 12-lead electrocardiogram (ECG) obtained on admission showed normal sinus rhythm, abnormal superior axis deviation, left ventricular hypertrophy and secondary repolarization changes (Fig 1A).

During her hospitalization, the patient developed episodes of sinus arrest with durations of up to 4.2 seconds, followed by bradycardia, frequent premature ventricular beats and repeated episodes of SVT at a rate of 160/min, the latter responsive to intravenous propranolol. A Holter monitor documented sinus bradycardia, junctional rhythm at a rate of 29, sinus arrest and SVT. A permanent A-V sequential demand pacemaker (manufactured by American Optical Co.) was implanted and the patient continued on propranolol and procainamide. For the following six months after hospitalization, she has remained relatively symptom free with only rare short-lived episodes of SVT.

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FIGURE 1A. Twelve lead surface ECG. B. High right atrial electrogram (AEG), His bundle electrogram (HBE), and leads 1, 2 and 3 of the surface ECG. On the His bundle electrogram tracing, atrial, His bundle and ventricular electrogram depolarizations are labelled A, H and V respectively.

MATERIALS AND METHODS

The patient was studied in the nonsedated post-absorptive state. Under local lidocaine anesthesia two 5F bipolar electrode catheters were introduced via the basilic vein and positioned at the high right atrium under fluoroscopic control for the purpose of both atrial pacing and intra-atrial electrogram recording.

A 6F tripolar catheter electrode was introduced via the femoral vein by Seldinger technique and positioned across the tricuspid valve. His bundle electrograms were recorded by the method of Scherlag and associates. Intra-atrial electrograms and leads 1, 2, 3 of the surface electrocardiogram were also monitored and recorded on an Electronics for Medicine DR-8 multichannel recorder at 100-200 mm/sec paper speed.

Rectangular pulses of 3 msec duration and 16 times diastolic threshold were used to stimulate the atrium at rates of 90 to 150/min. A-V refractory period (AVRP) measurements were obtained by use of a programmed specially constructed stimulator. This unit is capable of delivering a variably-coupled extra pulse ($S_2$) following every sixth basic ($S_1$) pulse.

RESULTS

During sinus rhythm A-H and H-V intervals, P wave morphology, activation sequence and conduction times were normal (Fig 1B). Utilizing the extrastimulus method an AVRP curve was constructed (Fig 2). Late diastolic coupled atrial premature depolarizations resulted in a single ventricular response with a normal morphology and no significant conduction delay (Fig 3A). Earlier coupled extrasystoles (Fig 3B) resulted in pronounced configurational changes of the ventricular response with prolongation of the H-V interval. This prolongation of intraventricular conduction as manifest by H-V prolongation and aberrantly conducted ventricular responses was found to be proportional to the extrasyntolic coupling interval (Fig 2).

The earliest coupled extrasystole elicited demonstrated marked intraventricular conduction delay and resulted in sustained SVT (Fig 4A). It was interesting to note that during the episode of SVT marked QRS configurational changes occurred. Initially, left bundle branch block with superior axis deviation was seen (Fig 4A) and subsequently right bundle branch block with marked right axis deviation was seen (Fig 4B).

DISCUSSION

Paroxysmal supraventricular tachycardia is a common arrhythmia not infrequently affecting young healthy persons. Among the mechanisms
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Figure 3A. Effect of late diastolic coupled atrial premature depolarization. Single ventricular response with normal configuration and no significant conduction delay is produced. Abbreviations are as in Figure 1. \( S_1S_2 \) is the coupling interval. B. Effect of early diastolic coupled atrial extrasystole. Marked change in QRS configuration and prolongation of H-V interval occurred.

Proposed to explain SVT two have been well defined and demonstrated in both in vitro and in vivo studies in the experimental animal,\(^5,6\) and the human being:\(^2\) (a) an ectopic atrial focus and (b) reentry of atrial impulses at the A-V junction. The presence of an atrial ectopic focus has been shown in the past to be the mechanism of SVT in studies by Scherf\(^10\) and Prinzmetal et al.\(^11\) Goldreyer and associates\(^3\) documented ectopic atrial tachycardia in three patients and commented about the features and therapeutic implications of separating this mechanism from reentry.

In the series reported by Goldreyer et al\(^{14}\) and Bigger and Goldreyer,\(^9\) atrial reentry utilizing the A-V conducting system was the explanation for recurrent episodes of induced or spontaneous SVT in all the patients studied. Reciprocal beating in the human heart was first reported by White in 1915.\(^12\) Rosenblueth,\(^13\) in 1958, suggested the presence of a dual A-V conducting pathway with a final common pathway located at the level of the bundle of His.

Figure 4A. Onset of SVT. Earliest coupled extrasystole produced the most marked intraventricular conduction delay (prolonged H-V interval) and changes in QRS configuration. This resulted in sustained SVT with QRS configuration resembling left bundle branch block with superior axis deviation. Ladder diagram illustrates time sequence in the His bundle tracing. \( A-A \) intervals, in msec, are shown in upper part of panel. HV intervals are shown in middle portion of panel and \( V-V \) intervals are listed in lower portion of diagram. See text for further discussion. B. Subsequent recordings during same episode of SVT. Note dramatic changes in QRS morphology with right bundle branch block configuration with right axis deviation.

Moe and Mendez\(^{14}\) more recently modified Rosenblueth's hypothesis on atrial reentry and demonstrated the presence of longitudinal dissociation in the upper part of the A-V junction. They postulated a dual A-V conducting pathway at that level as the physiologic setting for reentry. Moe et al\(^{14}\) have shown in open heart dogs and isolated rabbit heart preparations that the atrium is an essential link in the reentry pathway. Wallace et al\(^{15}\) have further emphasized that pathways necessary to produce atrial echoes do not extend below the A-V nodal level. Wit et al\(^{15}\) and Janse et al\(^{19}\) have demonstrated in vitro the presence of a circus movement along a reentrant pathway in sustained VT. James\(^{16}\) has recently defined a possible structural basis for reentry along the bundle of His, in both human and animal hearts. Han\(^{17}\) has suggested the possibility of a supraventricular impulse...
initiating ventricular ectopic impulses by reentry along a circuit involving both bundle branches.

In the case presented, induced late diastolic extrasystoles failed to reproduce SVT (Fig 3B). Only at critical coupling interval could a single atrial premature beat then induce pronounced prolongation of HV interval and intraventricular conduction (Fig 4A). This resulted in reentry of the ectopic impulse and subsequent sustained SVT. In part, this progressive HV prolongation resembles previously described properties of the human and canine A-V conduction system.\textsuperscript{8,16,18} However, in no prior instance has progressive HV prolongation been reported to result in sustained entry. Experimental evidence in the animal and human heart have demonstrated reentry to occur at the higher junctional tissues. The present case illustrates that the distal A-V conduction system may participate in a reentry circuit producing and perpetuating SVT. In the present study a distal site of reentry would suggest that this rhythm more appropriately be considered a variant of ventricular tachycardia.

### References


### Master Physiologist of the Eighteenth Century

The master physiologist of the century, a truly universal man, was Swiss-born Albrecht von Haller (1708-1777) who as a Wunderkind produced a Chaldean grammar, a Greek and Hebrew dictionary, poems, biographies. At the University of Göttingen he taught all branches of medicine, established botanical gardens and churches, wrote thousands of scientific papers. He became eminent as a botanist, anatomist, novelist, poet, public health official, founder of an orphan asylum, head of his native canton of Bern. He carried on a gigantic correspondence (some 14,000 letters) with many eminent figures of the enlightenment, ranging from Voltaire to the arch-lover Giovanni Casanova. Haller's finest work was done on the physiology of blood vessels and of the nervous system; he established that irritability is a property of muscles, while sensibility is characteristic of nerves; these observations correctly supported the myogenic theory of the heartbeat. He also established the role of bile in the digestion of fat and was a pioneer in the study of angiology and microscopic anatomy. His concept of physiology was that of anatomia animata, the science of biologic movement.

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